

Chapter 4

Diseases of the Porcine, Ovine, and Caprine Gastrointestinal Tract

Timothy H. Ogilvie

I. PORCINE GASTROINTESTINAL TRACT

- A.** Stomach. Gastric ulcers may be found at any production stage in swine.
1. Clinical findings include:
 - a. Sudden death resulting from perforation of a major blood vessel or acute, diffuse peritonitis
 - b. Melena
 - c. Anemia
 - d. Poor growth
 2. Etiology and pathogenesis. Risk factors for the development of gastric ulcers include finely ground feed, wheat feeds, and stressors (e.g., concurrent disease, competition for social status). Also, there has been an association made with vitamin E and selenium deficiency.
 3. Diagnostic plan. The diagnosis is often made on necropsy or through clinical signs.
 4. **Laboratory** tests confirm anemia and blood in the feces.
 5. Therapeutic plan. Treatment often is not carried out in individual animals. Affected animals may be culled. If warranted, treatment may follow similar courses as with other monogastrics (i.e., antacids, mucosal protectants, H₂-receptor blocking agents). Note that with H₂-receptor blocking agents, such as cimetidine, extra-label use is required.

B. Intestines

1. Diarrhea in neonatal swine
 - a. Enterotoxigenic *Escherichia coli* (ETEC)
 - (1) Patient profile and history. ETEC is a common cause of acute diarrhea in newborn piglets and can be a significant cause of diarrhea up to weaning age.
 - (2) Clinical findings. The condition presents as acute diarrhea (**nonhemorrhagic**) with dehydration, anorexia, weakness, and, in advanced cases, signs of shock and death caused by hypovolemia and acidemia.
 - (3) Etiology and pathogenesis
 - a. Many strains of ETEC are causative (more than 300 strains with varying combinations of O, K, and H antigens). Risk **factors** include poor hygiene, poor quality or insufficient colostrum, weak piglets at birth, and low herd immunity.
 - b. Route of infection. Enterotoxigenic strains of *E. coli* adhere to the mucosa of the small intestine, proliferate, and elaborate an enterotoxin, which causes the secretory diarrhea. Adherence to the small intestinal mucosa is achieved through any of several bacterial surface pili (K88, K99, 987P). Secretion is mediated through bacterial enterotoxins (heat-stable toxins, heat labile toxins, or both), which act on intact intestinal mucosa. These toxins increase the secretion of fluids and electrolytes to the gut lumen through activation of cyclic guanosine monophosphate (cGMP) or cyclic adenosine monophosphate (cAMP) systems. Absorptive capabilities of the **mucosal** cells remain **intact**.
 - (4) Diagnostic plan. Diagnosis is based on clinical signs, response to therapy, and laboratory confirmation.
 - (5) Laboratory tests include:
 - a. Fecal culture and *E. coli* identification using a pooled antigen antiserum

- (b) **Histopathology** of intestinal sections revealing gram-negative bacteria attached to relatively healthy-looking intestinal mucosa
- (6) **Therapeutic plan.** This condition is readily responsive if correct therapy is initiated early and encompasses the entire litter. Treatment includes:
 - (a) **Antibiotics** with a therapeutic index for gram-negative infections (e.g., trimethoprim-sulfas)
 - (b) Free-choice oral **electrolyte solutions**
- (7) **Prevention**
 - (a) **Management strategies.** Improvements in hygiene should be recommended and a system of "all-in, all-out" movement of swine groups implemented if possible.
 - (b) **Vaccination.** Immunity of sows (particularly gilts) may be improved through vaccination with an *E. coli* bacterin or early exposure to farrowing room flora.
 - (c) **Antibiotics** may be used prophylactically in litters during an outbreak.
- b. Transmissible gastroenteritis (TGE)**
 - (1) **Patient profile and history.** This disease, which is common in North America, presents in two forms: endemic (or mild) and epidemic. The epidemic form causes explosive outbreaks in neonatal piglets with high morbidity and mortality. The condition can be devastating to producers, both economically and psychologically.
 - (2) **Clinical findings**
 - (a) **Epidemic form.** There is acute diarrhea with vomiting, dehydration, and death.
 - (b) **Endemic form.** There is chronic diarrhea late in the nursing period or in the weaner pig population. Although mortality is low, growth retardation is significant. Diarrhea is sporadic.
 - (3) **Etiology and pathogenesis.** The epidemic and endemic forms are caused by separate and distinct **coronaviruses**.
 - (a) The **epidemic form** results from severe enteric damage caused by the coronavirus. There is villus atrophy, resulting in compromised gut function, malabsorptive and osmotic diarrhea, severe dehydration, and death. If death does not ensue, piglets recover as the enterocytes mature to cover the villus (5–7 days).
 - (b) With the **endemic form**, the pathogenesis is similar to the epidemic form but less pronounced.
 - (4) **Diagnostic plan.** The diagnosis is aided by clinical findings, infection profile, and unresponsiveness to therapy. Laboratory tests are confirmatory.
 - (5) **Laboratory tests** include intestinal histopathology, virus isolation from the feces or intestine, immunofluorescence of tissue, and serology.
 - (6) **Therapeutic plan.** Neither form of TGE is responsive to antibiotics or supportive treatment.
 - (7) **Prognosis**
 - (a) **Mortality.** With the epidemic form, virtually all piglets die within a one-month age cohort (2 weeks prepartum to 2 weeks old at the time of infection).
 - (b) **Normal production** resumes approximately 1 month after infection.
 - (c) **Herd immune response** may be hastened by feeding the fecal content of infected pigs to sows who have yet to produce colostrum and farrow.
 - (8) **Prevention**
 - (a) **Management strategies.** The herd should be closed and new introductions farrowed offsite. Biosecurity prevents the entry of pigs or fomites (e.g., birds, rodents; visitors). The virus may disappear over time.
 - (b) **Vaccination** may be beneficial in an area where prevalence is high. Both killed and modified live virus vaccines are available.
- c. Rotavirus.** This condition is similar to the endemic form of TGE (see 1B 1 b). Rotavirus infection in pigs is similar to the same infection in other species (see Chapters 2 and 3). Vaccination lessens the effect of infection.
- d. Hemagglutinating encephalomyelitis virus (HEV, vomiting and wasting disease)**

- (1) **Patient profile and history.** This disease produces vomiting and weight loss in nursing pigs. There may be acute encephalomyelitis. Infection is global, as evidenced by serological findings. Outbreaks occur with high morbidity and mortality.
- (2) **Clinical findings.** Vomiting is the major gastrointestinal tract sign followed by emaciation and dehydration. Diarrhea is not usually a finding. Wasting, over time, is followed by death.
- (3) **Etiology and pathogenesis.** The condition is caused by a coronavirus. In the case of encephalomyelitis, the virus may be isolated from the central nervous system. The pathogenesis of the vomiting and wasting component is unclear. There may be a central cause for the gastrointestinal tract signs or a local effect of the virus on the stomach.
- (4) **Diagnostic and therapeutic plans.** The diagnosis is based on histopathologic findings. There is no treatment for this condition.
- (5) **Prevention.** The disease runs its course in 2–3 weeks, and herd immunity is good.
- e. Coccidiosis**
 - (1) **Patient profile and history.** Coccidiosis is common in young pigs. It is a disease of high morbidity and low mortality.
 - (2) **Clinical findings.** Commonly, a profuse diarrhea occurs after piglets are 1 week of age. There is no blood or mucus evident. Piglets may become anorexic and dehydrated. The diarrhea may persist for 3–5 days, and within a herd, the course may be chronic where there is continuous movement of animals on and off the premises.
 - (3) **Etiology and pathogenesis.** *Isospora suis* is the causative coccidian parasite. Similar to other coccidia, sexual and asexual life cycles occur in the intestinal tract. Asexual reproduction in the small intestinal mucosa produces villus atrophy and destruction of villus epithelial cells.
 - (4) **Diagnostic plan.** Diagnosis depends on the laboratory findings.
 - (5) **Laboratory tests** include the demonstration of significant numbers of oocytes in the feces, histopathology of the small intestine, direct smears of intestinal contents, or combinations of these techniques. Diarrhea may occur before patency of infection, during biphasic peaks of patency, or after oocyte production has waned. Consequently, detection of oocysts may be difficult and require necropsy confirmation, evaluation of the feces of littermates, or both.
 - (6) **Therapeutic plan.** Treatment during clinical disease is of little value.
 - (7) **Prevention**
 - (a) **Environmental conditions.** The infection creates most problems under conditions that allow for environmental contamination and transfer of infective oocysts. Oocysts are difficult to kill. Housing pigs on dirt or cracked concrete floors provides suitable conditions for maintenance of the organism in the environment. Wet floors encourage chilling of the piglet and infection by oocysts.
 - (b) **Preventive procedures** include maintenance of a dry environment, raising piglets on slatted floors to reduce the fecal–oral spread, or resurfacing floors to eliminate cracks.
- f. Clostridium perfringens enteritis**
 - (1) **Patient profile and history.** This condition is seen most commonly in nursing pigs but may occur in animals up to 10 weeks of age. The disease is most common in western North America.
 - (2) **Clinical findings.** *C. perfringens* type C produces an acute, hemorrhagic diarrhea in suckling pigs, which rapidly results in death. In older (even weaned) pigs, a more protracted, nonbloody diarrhea is found and results in a lower mortality. *C. perfringens* type A has been reported to produce diarrhea and poor growth rates.
 - (3) **Etiology and pathogenesis.** The clostridial organisms are soil borne and enter the animal through the oral route. Attachment to the intestine and proliferation occurs with the production of a β -toxin (*C. perfringens* type C), which is

responsible for the necrosis of the intestinal mucosa and the development of clinical signs.

- (4) **Diagnostic plan.** Diagnosis is based on laboratory findings.
- (5) **Laboratory tests** include necropsy findings, histopathology, and intestinal tract smears. Culture of intestinal contents is inconclusive because *Clostridia* make up much of the natural gut flora.
- (6) **Therapeutic plan.** Treatment is unrewarding and should be reserved for very early cases. Penicillins may be used.
- (7) **Prevention.** Vaccination with a type-specific or multi-spectrum toxoid is recommended.

2. Diarrhea in feeder pigs

a. Swine dysentery

- (1) **Patient profile and history.** This disease is endemic in many operations and very costly to the economic production of pork. It is most common in the 7- to 16-week age group.
- (2) **Clinical findings.** There is a mucohemorrhagic enteritis with anorexia, dehydration, and poor growth rates in affected pigs. The disease may be of high morbidity and moderate mortality. The condition may last 3-4 weeks in untreated animals. A chronic diarrhea may result in some pigs.
- (3) **Etiology and pathogenesis.** The causative organism is *Serpulina hyodysenteriae*. It invades colonic crypts and produces an erosive colitis, resulting in colonic malabsorption, diarrhea, dysentery, and the mucoid feces seen clinically.
- (4) **Diagnostic plan.** The diagnosis is confirmed by laboratory findings.
- (5) **Laboratory tests** include dark field microscopy or stained fecal smears, fecal culture, and identification via fluorescent antibody staining or slide agglutination. Serological tests may aid in the identification of carrier pigs.
- (6) **Differential diagnoses** include salmonellosis, proliferative enteritis, spirochetal diarrhea, and hog cholera.
- (7) **Therapeutic plan**
 - (a) Effective treatments include any of the following drugs at labeled dosages and routes of administration. However, relapses may occur. Treatment is usually administered to all animals in the group by water medication. Individual animals may require selective treatment if dehydrated and anorexic.
 - (b) **Medications** include tylosin, dimetridazole, ronidazole, lincomycin, tiamulin, and carbadox.
- (8) **Prevention.** If the disease is present, there are two management responses.
 - (a) Live with the condition through continuous medication to control the clinical expression.
 - (b) Eradicate the organism through depopulation and repopulation. **Depopulation** may be carried out in two ways:
 - (i) One radical method includes the removal of all pigs coupled with disinfection of the premises and restocking with known disease-free hogs.
 - (ii) All pigs may be mass-medicated simultaneously with premise disinfection, serial depopulation of pigs, and restocking with disease-free pigs.
 - (c) **Management strategies.** Either system will eliminate the disease but must be followed by fastidious hygiene and biosecurity to restrict the entrance of the organism via carrier pigs, rodents, or manure-contaminated fomites.

b. Spirochetal diarrhea. This condition is similar to swine dysentery but may be found in clean herds. It is caused by an unknown spirochete (*Spirochaeta innocens* variety). The infection produces a mild postweaning diarrhea with weight loss. It responds to medication in a way similar to swine dysentery [see I B 2 a (7)].

c. Nonspecific colitis. This condition is also similar to swine dysentery but is not associated with a demonstrable pathogen (see I B 2 a). This condition may be associated with high-protein or pelleted feeds and may respond to a feed change.

d. Postweaning coliform gastroenteritis

- (1) **Patient profile and history.** This condition affects pigs from a few days to 2 weeks post weaning. Morbidity may approach 100%, but the mortality is usually moderate.
- (2) **Clinical findings.** A few pigs may be found dead, and within a few days, most remaining pigs in the group are showing signs of diarrhea, skin discoloration, anorexia, and a marked loss of condition. The course of the disease is usually 7-10 days.
- (3) **Etiology and pathogenesis**
 - (a) The condition is caused by one of several serotypes of **ETEC**. Colonization and proliferation of the *E. coli* in the small intestine produces the clinical signs.
 - (b) The **pathophysiological development** of diarrhea is similar to ETEC in neonatal piglets [see I B 1 a (3)]. However, there are a variety of associated **risk factors** for this condition including:
 - (i) Concurrent infection with rotavirus
 - (ii) Stressors (mixing, weaning)
 - (iii) Loss of lactogenic immunity
 - (iv) Normal intestinal villus turnover
- (4) **Diagnostic plan and laboratory tests.** Necropsy results coupled with intestinal tract culture findings are confirmatory.
- (5) **Differential diagnoses** include salmonellosis and swine dysentery.
- (6) **Therapeutic plan.** Mass medication of all pigs in the group is essential. Water medication is best with one of many broad-spectrum antimicrobials. Electrolytes should be offered in the drinking water.
- (7) **Prevention.** Recommendations for control are empirical and unproven, although many producers will prophylactically medicate growing pigs and introduce feed changes or other sources of stress gradually.

e. Salmonellosis

- (1) **Patient profile and history.** This disease may occur in a septicemic or enteric form.
- (2) **Clinical findings**
 - (a) With **septicemia**, there is sudden death or **terminal signs** of septicemia including cyanosis, subcutaneous petechial hemorrhages, and recumbency with convulsions.
 - (b) The **enteric form** presents as diarrhea with occasional signs of pneumonia or encephalitis. A chronic diarrhea with resultant poor growth, rectal strictures, or both may develop.
- (3) **Etiology and pathogenesis.** The septicemic form is most commonly caused by *Salmonella choleraesuis*, whereas the enteric form yields cultures of *S. typhimurium*. The pathogenesis is as for other species with salmonellosis (see Chapters 2 and 3).
- (4) **Diagnostic plan and laboratory tests**
 - (a) The **septicemic form** is almost invariably fatal, and diagnosis is based on necropsy, histopathology of intestinal tissue, and culture and sensitivity of intestinal contents and tissues (e.g., lymph nodes).
 - (b) The **enteric form** is diagnosed on postmortem. Culture of the feces of live pigs may be attempted but is subject to the same drawbacks as fecal culture in horses or ruminants.
- (5) **Therapeutic plan.** Enteric or broad-spectrum antimicrobials may be used with limited success even in the enteric form.
- (6) **Prevention.** The occurrence of the disease is lessened by attention to hygiene and group movements of pigs through barns to allow for periodic disinfection of premises.

f. Porcine proliferative enteropathy (PPE)

- (1) **Patient profile and history.** This condition affects pigs from weaner age through to young adults. As with other disorders, this condition affects hogs that are intensively raised in confinement. This condition may be seen more often in minimal disease (specific pathogen-free) facilities and appears worldwide.

- (2) **Clinical findings**
 - (a) There may be **sudden death** and **hemorrhagic diarrhea** in older feeder pigs (gilts and boars). This manifestation of the disease is called **porcine hemorrhagic enteropathy**.
 - (b) PPE is most commonly seen as diarrhea, anorexia, and ill thrift in growing pigs. Pigs are afebrile but suffer from chronic, intermittent diarrhea.
 - (3) **Etiology and pathogenesis.** Older literature associates the condition with *Campylobacter mucosalis*. The clinical syndromes are now associated with a new pathogen, *Lawsonia intracellularis*. The pathogenesis is not well understood, but affected animals develop a regional ileitis, hypertrophic terminal ileum, and hemorrhagic and necrotic enteritis with the passage of blood in acute severe cases.
 - (4) **Diagnostic plan and differential diagnoses.** Clinical findings are helpful in differentiating this condition from postweaning coliform gastroenteritis. Postmortem examination is the method of choice for diagnosis.
 - (5) **Therapeutic plan.** Treatment is often not undertaken in individual animals but could be attempted by using macrolides (lincomycin, tylosin). Control with medicated feeds and management changes is variable.
- g. Intestinal parasites.** Enteric parasites are common and impact productivity in feeder barns.
- (1) ***Trichuris suis***
 - (a) **Clinical findings.** Clinically, the signs of swine **whipworm** infestation may appear similar to swine dysentery (see 1B 2 a).
 - (b) **Diagnostic plan.** Diagnosis is by fecal floatation of worm eggs and by postmortem evaluation.
 - (c) **Therapeutic plan.** Dichlorvos anthelmintics are therapeutic.
 - (d) **Prevention.** Eggs are very resistant in the environment, and attention to hygiene is necessary to prevent ongoing infestations.
 - (2) ***Ascaris suum***
 - (a) **Patient profile and clinical findings.** Roundworms are common in growing pigs. They cause growth impairment at high infestation rates and are associated with some cases of respiratory disease caused by the larval migration patterns through the lungs. The larvae also produce "white-spotted liver lesions" caused by hepatic migration during maturation.
 - (b) **Diagnostic plan.** Diagnosis is based on fecal floatation findings or slaughter checks of viscera (liver).
 - (c) **Therapeutic plan and prevention.** Treatment and control is based on hygiene and regular, strategic anthelmintic usage: sows 7 days before farrowing, and feeder pigs at 50 kg or every 3 weeks. Improvements in hygiene limit direct fecal-oral exposure of infective eggs.
 - (3) ***Hyostrongylus rubidus***
 - (a) **Patient profile and etiology.** *Hyostrongylus* causes anemia, poor growth, and diarrhea in feeder pigs because of a parasitic gastritis. In older animals, this worm has been associated with "thin sow syndrome."
 - (b) **Clinical findings.** Animals may carry heavy infestations without showing clinical signs.
 - (c) **Diagnostic plan.** Diagnosis is made by clinical findings and fecal egg identification.
 - (d) **Therapeutic plan and prevention.** If this parasite is suspected of causing problems, regular deworming with levamisole or dichlorvos is warranted.
 - (4) ***Oesophagostomum***
 - (a) **Patient profile.** This is the nodular worm of the large intestine that produces inflammatory nodules in the colon, cecum, and rectum.
 - (b) **Clinical findings.** There are few clinical signs expressed with infestation unless concurrent infections with intestinal pathogens occur (e.g., *Salmonella* species).
 - (c) **Diagnostic plan.** Diagnosis is based on necropsy or fecal floatation.
 - (d) **Prevention** of this and other gastrointestinal parasites include improving hygiene, removing manure, feeding above floor level, and other ways of limiting the fecal-oral spread of parasite eggs.

II. OVINE AND CAPRINE GASTROINTESTINAL TRACT

A. Diarrhea

1. **Salmonellosis**
 - a. **Patient profile and history.** This disease is seen in young lambs or adults with a recent history of stress (e.g., shipping, parturition).
 - b. **Clinical findings.** There may be a sudden onset of fever, depression, and diarrhea with fibrin and blood. Abortion may be seen within the flock.
 - c. **Etiology and pathogenesis.** *Salmonella dublin* or *S. typhimurium* are usually isolated. Pathogenesis is similar to other species of host (see Chapters 2 and 3).
 - d. **Diagnostic plan, therapeutic plan, and prevention.** These plans are identical to those in other species (see Chapters 2 and 3).
2. ***Clostridium perfringens* type C**
 - a. **Patient profile.** This infection occurs in lambs and kids less than 3 weeks of age.
 - b. **Clinical findings and etiology.** Proliferation of *C. perfringens* type C in the intestine causes elaboration of a β -toxin, which causes an acute hemorrhagic enteritis in affected animals.
 - c. **Diagnostic plan, therapeutic plan, and prevention.** These plans are similar to those of the neonatal calf (see Chapter 3).
3. ***Clostridium perfringens* type D (pulpy kidney disease, overeating disease, enterotoxemia)**
 - a. **Patient profile.** This infection is seen in young, rapidly growing animals on a high plane of nutrition.
 - b. **Clinical findings.** Various clinical pictures are described, but clinical signs are apparent only early in the course of the disease.
 - (1) **Sudden death** is the most common presentation.
 - (2) **Neurological signs** are observed before death.
 - (3) **Transient diarrhea in sheep** occurs early in the course of the fatal disease; diarrhea may be a finding.
 - (4) **Persistent diarrhea in goats** is a specifically different syndrome of clostridial enterotoxemia. Diarrhea is chronic with weight loss. Feces contain flecks of mucus and blood.
 - c. **Etiology and pathogenesis**
 - (1) **Sheep.** Pulpy kidney disease results from the proliferation of *C. perfringens* type D in the small intestine. The bacteria release a number of toxins, including the ϵ toxin. This toxin causes vascular and nervous system damage.
 - (2) **Dairy goats** are often fed a high-energy ration during lactation, predisposing them to intestinal clostridiosis. This condition may take the form of acute disease with sudden death or produce a more chronic diarrhea and wasting form of disease.
 - d. **Diagnostic plan and laboratory tests**
 - (1) **Sheep.** In lambs, the history, clinical findings, and necropsy findings are diagnostic. In adult sheep, pulpy kidney disease must be differentiated from rabies, acute lead poisoning, hypomagnesemic tetany, pregnancy toxemia, and loup-ill (see Chapter 11).
 - (2) **Goats.** Fecal collection for toxin identification (mouse bioassay) is necessary for accurate diagnosis.
 - e. **Therapeutic plan**
 - (1) **Feed.** Treatment is centered on decreasing the energy component of the ration. This advice may run counter to the objectives of the owner.
 - (2) **Vaccination** with *C. perfringens* type D toxoid is effective in decreasing the severity of the disease but does not eliminate it in the face of high-energy rations. Vaccination every 6 months may be warranted. Local reactions occur at the vaccination sites.
4. ***Clostridium septicum* (braxy)**
 - a. **Patient profile.** This infection occurs in sheep sporadically in North America.

- b. **Clinical findings.** Sudden death is the most common presentation. Early signs include anorexia, fever, abdominal pain, and distention.
 - c. **Etiology and pathogenesis.** Braxy is an infectious abomasitis-toxemia of sheep caused by *C. septicum*. Bacteria or spores are introduced through breaks in the abomasal mucosa, proliferate and cause death through the systemic absorption of toxins. Infection occurs only in the winter months and is associated with consumption of frozen feeds.
 - d. **Diagnostic plan.** The diagnosis is made by isolating *C. septicum* from the typical inflammatory lesions of the abomasum or from necropsy specimens.
5. **Intestinal helminthiasis**
- a. **Patient profile.** This condition is common in sheep and should not be overlooked or underemphasized. Lambs and kids are affected more than adults.
 - b. **Clinical findings**
 - (1) The **clinical hallmarks** of infestation are weight loss, poor growth, diarrhea, and edema.
 - (2) **Other signs** include poor feed conversion, wasting, and eventually death of the host. Sudden death may be the presenting problem with *Haemonchus contortus* infestation. Not all parasites produce the entire range of clinical signs.
 - (3) *Haemonchus* and *Bunostomum* species feed off of blood with resultant anemia.
 - c. **Etiology and pathogenesis.** Infective eggs and larvae are ingested usually at pasture and mature in various areas of the gastrointestinal tract. An exception to this is the tapeworm, which requires an intermediate host in their life cycle. This host (an oribatid mite) is ingested by the ruminant. The total effect of a heavy parasite burden is more significant in the young than in the mature animal.
 - (1) **Abomasum**—*Haemonchus contortus*, *Ostertagia circumcincta* and *O. trifurcata*, *Trichostrongylus axei*
 - (2) **Small Intestine**—*Trichostrongylus* species, *Nematodirus* species, *Bunostomum* species, *Cooperia* species, *Strongyloides papillosus*, *Moniezia* species, *Thysanosoma* species
 - (3) **Large Intestine**—*Oesophagostomum* species, *Chabertia ovina*, *Trichuris* species
 - d. **Diagnostic plan and laboratory tests.** Diagnosis depends on the demonstration of fecal larvae, fecal flotations and egg counts, necropsy, and intestinal worm counts.
 - e. **Therapeutic plan and prevention.** Parasite control strategies for most sheep-rearing areas in North America include:
 - (1) **Prelambing treatment.** This treatment of ewes is critical to prevent the occurrence of a periparturient rise in the fecal egg counts. Treatment with a product effective against hypobiotic larvae is essential (e.g., levamisole, ivermectin). The usual management strategy dictates a winter treatment in northern climates to prevent maturation of worms and eventual seeding down of the pastures in the spring.
 - (2) **Creation of safe pastures**
 - (a) Lambs **raised indoors** in a relatively parasite-free environment may be turned out onto virgin pastures without anthelmintic treatment. The only indoor risk of infestation is from *Coccidia* or *Strongyloides papillosus*.
 - (b) **Regrowth pasture** after harvested crops generally can be considered as safe. Pastures previously grazed by cattle or other species are generally safe for sheep and goats because of the limited cross-infection between species.
 - (3) **Deworming.** In the spring, deworming of animals prevents the summer buildup of parasite eggs and larvae. A regimen of 4–8 treatments should be considered at 3-week intervals throughout spring and summer.
 - (4) **Treat and move.** A single treatment followed by movement to a safe pasture limits reinfestation.
 - (5) **Anthelmintic agents**
 - (a) **Levamisole** given at 7.5 mg/kg orally and **ivermectin** given at 0.2 mg/kg orally are highly efficacious for the control of sheep parasites.

- (b) **Thiabendazole**, **piperazine**, and **phenothiazine** may still be used but have a narrower spectrum of activity or suffer from parasite resistance patterns.

6. Coccidiosis

- a. **Patient profile.** Coccidiosis can occur in any sheep or goat but is most significant in the young. Clinical signs are not expressed in animals until 2–3 weeks of age and are most common in lambs 1–4 months of age.
- b. **Clinical findings.** There is a severe, watery diarrhea that lasts for 1–2 weeks. The diarrhea may be hemorrhagic. Also evident are dehydration, weakness, anorexia, and death as the disease progresses.
- c. **Etiology and pathogenesis.** *Eimeria arloingi* and *E. ovina* are the most commonly diagnosed coccidial pathogens. The pathogenesis is similar to bovine coccidiosis (see Chapter 3).
- d. **Laboratory tests.** The diagnosis is confirmed by the demonstration of large numbers of *Coccidia* oocysts in the feces. Also, lambs will be hypoproteinemic and anemic.
- e. **Therapeutic plan and prevention**
 - (1) **Sulfamethazine** at 30 mg/kg daily is added to the drinking water. Lambs and kids are treated at 3–4 weeks of age for 7 days. This is repeated in 3 weeks. Treatment may need to continue in 3-week cycles until weaning in some flocks or herds.
 - (2) **Monensin** may be added to the ration at 5–7.5 g/ton of total pelleted ration. Feeding of this medicated ration should continue until 3 weeks post weaning.
 - (3) **Lasalocid** is effective at 25 mg/kg of feed or 2–5 mg/kg body weight/day. It may also be fed as a ground salt at .75%.
 - (4) **Amprolium** may be used on kids or lambs 2 weeks of age at 50–60 mg/kg for 10 days. Thiamine deficiency may result from the use of this product (see Chapter 11).
 - (5) **Decoquinat** is an excellent preventive and may be mixed at home. It should be used at 5 mg/kg. It should be introduced at 2 weeks of age and fed for 1 month or longer. Older animals may be offered salt with 2 kg of 6% decoquinat premix in 45 kg of salt.
- 7. **Toxicities.** As with cattle, toxic agents (e.g., lead arsenate, copper sulfate, superphosphate fertilizers) may cause gastrointestinal signs (see Chapter 3).
- 8. **Dietary disturbances.** Nutritional diarrheas are common in lambs turned out onto lush pastures or introduced to high-energy rations. These conditions (mainly diarrheas) are self-limiting and are prevented by more gradual introduction to the ration. Occasionally, symptomatic treatment may be necessary if the diarrhea is severe.

- B. Chronic weight loss** in sheep or goats may result from the involvement of any one of several body systems. Differential diagnoses include: primary undernutrition, secondary undernutrition, parasites (ectoparasites and endoparasites), infectious diseases (Johne's disease, pseudotuberculosis, chronic pulmonary disease, chronic degenerative joint disease), chronic renal disease, other chronic disease.

1. Paratuberculosis (Johne's disease)

- a. **Patient profile and history.** Goats are affected more commonly than sheep and are usually 1 year of age or older before showing clinical signs. The onset of clinical signs coincides with stress (parturition, heavy lactation, environmental or management changes).
- b. **Clinical findings.** Animals with this disorder exhibit chronic, progressive weight loss, a capricious appetite, and lethargy and weakness. The haircoat or wool is poor and breaks easily. Diarrhea and submandibular edema may occur terminally.
- c. **Etiology and pathogenesis.** *Mycobacterium paratuberculosis* is the causative organism. Transmission is oral, and ingested organisms localize in the gastrointestinal tract and adjacent lymph nodes. The organisms may remain dormant for extended periods of time.
- d. **Diagnostic plan** depends on the laboratory findings.
- e. **Laboratory tests**

- (1) There is **hypoproteinemia** and **hypoalbuminemia** along with a mild anemia and hypocalcemia. It is also common to find a concurrent heavy parasite burden on **fecal floatation examination**.
 - (2) **Reliable tests.** Many diagnostic tests suffer the same deficiencies as in the bovine (see Chapter 3). However, there are some reliable tests.
 - (a) **Fecal culture** is the most reliable indicator of infection.
 - (b) In the goat, the **agar gel immunodiffusion (AGID) test** has a sensitivity that approaches fecal culture and is a useful test for subclinical infection.
 - (c) Cold or modified complement fixation tests are the **serological tests** of choice in sheep.
 - (3) **Histological examination** of mesenteric and intestinal lymph nodes of affected sheep with acid fast stains are diagnostic.
 - f. **Therapeutic plan and prevention.** Treatment is impractical, and elimination efforts revolve around depopulation, either complete or selective via fecal culture. Prevention requires maintenance of a closed herd or flock.
2. **Visceral caseous lymphadenitis (CLA)**
- a. **Patient profile and history.** Goats and sheep are subject to this disease, and there may be a history of recent shearing or dipping (sheep) or other recent animal contacts. Draining abscesses may also be evident in the flock.
 - b. **Clinical findings**
 - (1) **Superficial lymph node abscess** may be found in the case under examination or in the flock (see Chapter 16 V D 2). In the case of visceral CLA, there are no pathognomonic clinical signs, simply insidious and progressive **weight loss**. Emaciation may occur over weeks or months and may not be visible on heavily fleeced or haired animals. Body surface palpation may be necessary to adequately evaluate flesh cover.
 - (2) **Concurrent bloat, dysphagia, or vagal indigestion** may be seen. Other body systems may be involved with signs referable to the respiratory tract, central nervous system (CNS), or mammary gland.
 - c. **Etiology and pathogenesis.** *Corynebacterium pseudotuberculosis* is the causative organism and resides in manure and soil. The gram-positive organism invades the body through both abraded and intact skin. It migrates and causes abscesses in both deep and superficial lymph nodes. Superficial lymph node abscesses, if present, are readily visible and palpable.
 - d. **Diagnostic plan and laboratory tests.** Visceral caseous lymphadenitis is difficult to diagnose. Laboratory findings that are helpful in defining the condition include:
 - (1) A possible leukocytosis with a normal lymphocyte to neutrophil ratio
 - (2) A possible hyperfibrinogenemia and hypergammaglobulinemia
 - (3) Hypoproteinemia secondary to decreased appetite and possible malabsorption
 - (4) Recovery of organism or signs of chronic peritonitis on abdominocentesis
 - (5) Detection of serum antibodies on paired sera submitted for enzyme-linked immunosorbent assay (ELISA)
 - e. **Therapeutic plan and prevention.** Exploratory abdominal surgery may be attempted in a valuable animal but is of limited value. Control is as for CLA (see Chapter 16 V D 2 f).
3. **Abomasal emptying defect in Suffolk sheep**
- a. **Patient profile.** This condition is seen specifically in Suffolk sheep and is hypothesized to be breed-related.
 - b. **Clinical findings** include anorexia, chronic weight loss, and abdominal and abomasal distension.
 - c. **Therapeutic plan and prevention.** Treatment is ineffective, and there are no recommended preventive procedures.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. Salmonellosis and pulpy kidney disease of lambs are similar in what way?
 - (1) There may be a sudden onset of diarrhea.
 - (2) Sudden death is the most common presentation.
 - (3) Chronic diarrhea with flecks of blood is a common presentation.
 - (4) Diagnosis is based on fecal culture.
 - (5) Eating frozen feeds increases the risk of disease.
2. Which statement regarding intestinal nematode parasites of sheep is true?
 - (1) They are of greatest clinical importance in adults.
 - (2) They cause the most problems in animals raised indoors or in feedlots.
 - (3) They may cause anemia and diarrhea.
 - (4) They are ingested as mature, egg-laying adults.
 - (5) They live primarily in the large intestine.
3. Which of the following statements regarding porcine proliferative enteropathy (PPE) is true? PPE:
 - (1) is seen most often in association with pneumonia.
 - (2) is a congenital disease in piglets.
 - (3) causes sudden death and encephalitis.
 - (4) is diagnosed best on postmortem examination of clinically diseased pigs.
 - (5) occurs in range pigs with heavy parasite burdens.
4. Which one of the following statements regarding visceral caseous lymphadenitis (CLA) is true?
 - (1) It affects only the gastrointestinal system.
 - (2) It is transmitted mainly through aerosolization.
 - (3) It may produce a hyperfibrinogenemia.
 - (4) It is restricted to sheep.
 - (5) It is caused by *Mycobacterium paratuberculosis*.
5. Which statement regarding gastric ulcer in swine is correct? Gastric ulcers in swine:
 - (1) are seen only in adult pigs.
 - (2) are most common in free-ranging pigs.
 - (3) are more common with coarse diets.
 - (4) may present with signs of sudden death or poor growth.
 - (5) cause diarrhea.
6. Which statement describing coliform bacteria that cause diarrhea in neonatal pigs is true?
 - (1) They are usually from a single strain.
 - (2) They elaborate an enterotoxin.
 - (3) They disrupt absorption capabilities of the small intestine.
 - (4) They cause a hemorrhagic diarrhea.
 - (5) They do not attach to enterocytes.
7. Transmissible gastroenteritis (TGE) and hemagglutinating encephalomyelitis (HEV) disease in swine differ in what way?
 - (1) TGE causes diarrhea, whereas HEV presents as vomiting and emaciation.
 - (2) Morbidity and mortality due to HEV infection is low compared to TGE.
 - (3) HEV occurs in weaner pigs, whereas TGE is a disease of nursing pigs.
 - (4) TGE is caused by a coronavirus and HEV, by a rotavirus.
 - (5) HEV is responsive to treatment, whereas TGE is not.
8. Coccidiosis causes which one of the following clinical signs?
 - (1) Bloody diarrhea in market age pigs
 - (2) Bloody diarrhea in nursing piglets
 - (3) Few clinical signs in swine
 - (4) Little morphologic change to intestinal cells
 - (5) Profuse diarrhea in young pigs

9. Successful management strategies to limit the production losses due to *Serpulina hyodysenteriae* include which of the following herd procedures?

- (1) One week of oral antibiotics followed by increased attention to biosecurity of the premises
- (2) Depopulation followed by premise disinfection and restocking with disease free stock
- (3) Ten days of mass water medication followed by oral electrolytes
- (4) Individual medication of sick animals followed by increased biosecurity of the premises
- (5) Decreased fiber in the diet followed by medication of any remaining sick animals

ANSWERS AND EXPLANATIONS

1. **The answer is 1** [I A 1, 3]. Both salmonellosis and pulpy kidney disease cause diarrhea, but pulpy kidney disease causes this only early in disease because death soon ensues. Sudden death is not a common feature of salmonellosis. Chronic diarrhea may occur with salmonellosis or in goats with clostridial enterotoxemia but not with pulpy kidney disease. Fecal culture of *Salmonella* organisms is disappointing because of the dilution factor associated with the diarrhea and because the organism attaches to the enterocyte. Toxin analysis, not bacteriology, is necessary to diagnose pulpy kidney disease. Frozen feed consumption is associated with braxy.

2. **The answer is 3** [I A 5 b]. Intestinal parasites in sheep may cause anemia, poor growth, diarrhea, edema, and weight loss. Parasites are of greatest clinical significance in young animals raised on pasture where infectious eggs or immature larvae are ingested off of or near blades of grass. Parasites may be harbored throughout the gastrointestinal tract.

3. **The answer is 4** [I B 2 f (4)]. Porcine proliferative enteropathy (PPE) affects pigs after weaning and is best diagnosed by sacrificing affected pigs for necropsy. Pigs confined to minimal disease (specific pathogen-free) facilities are most commonly affected. Pathology includes a regional ileitis and hypertrophic terminal ileum, resulting in the clinical signs of diarrhea and poor growth performance.

4. **The answer is 3** [I B 2 d]. Clinical pathology often reveals a hyperfibrinogenemia with this deep-seated infection. Other systems (e.g., respiratory) may be affected by the enlargement and abscessation of visceral lymph nodes. The organism (*Corynebacterium pseudotuberculosis*) is transmitted mainly through skin trauma of goats and sheep. *Mycobacterium paratuberculosis* is the causative agent of Johne's disease.

5. **The answer is 4** [I A 1]. Clinical signs with gastric ulceration in swine are melena, sudden death, anemia, or poor growth rates. Diarrhea is not a common finding. Risk factors include finely ground diets and crowded conditions. Growing pigs are at greatest risk.

6. **The answer is 2** [I B 1 a (3) (b)]. Enterotoxigenic *Escherichia coli* (ETEC) elaborate an enterotoxin that causes gut mucosa cells to secrete excess fluid and electrolytes. However, absorptive characteristics of the cells are maintained. There are many pathogenic stains of this organism that adhere to the enterocytes by bacterial surface pili. The diarrhea may be frothy, white, or brown but is not hemorrhagic.

7. **The answer is 1** [I B 1 b (2), d (2)]. Both transmissible gastroenteritis (TGE) and hemagglutinating encephalomyelitis virus disease (HEV) are diseases of high morbidity and high mortality. Both occur in nursing pigs, although TGE may occur in weaner pigs as well. Both are caused by coronaviruses, and neither is responsive to treatment.

8. **The answer is 5** [I B 1 e (2)]. Coccidiosis (infection with *Isospora suis*) causes a profuse diarrhea in young pigs. The diarrhea is not bloody, although the coccidian parasites cause atrophy and destruction of villus epithelial cells.

9. **The answer is 2** [I B 2 a (8)]. *Serpulina hyodysenteriae* is the causative organism of swine dysentery. Correct management procedures to limit the effects of disease in an infected population include continuous mass medication of all pigs or depopulation followed by disinfection of the premises and restocking with disease-free animals. This must then be followed by increased attention to hygiene and biosecurity measures to prevent re-introduction of disease.